Figure 4 show the nucleic acid sequence of the full length cDNA25 (SEQ ID NO: 26). The start and stop codons are underlined.

Figure 5A shows amino acid sequence of the full length cDNA25 (SEQ ID NO: 27). The antigenic peptide is underlined.

Please delete the paragraphs on page 7, line 31 through page 8, line 14.

Please replace the paragraph on page 92, line 23 through page 93, line 22 with the following:

Peptides were synthesized by a solid phase method using a peptide synthesizer (model AMS 422;Gilson Co.Inc., Worthington, OH)(>90% purity). The peptides to be synthesized were selected from the reported human sequence of gp100 based on HLA-A2.1 binding motifs (Falk, K., (1991) *Nature* 351:290; Hunt, D. F., et al, (1992) *Science* 255:1261; Ruppert, J., et al., (1993) *Cell* 74:929; Kubo, RT, et al. (1994) *J Immunol.* 152:3913). The following peptides were tested: Eight 8-mer peptides (with residues starting at -199, 212, 218, 237, 266, 267, 268, 269, eighty-four 9-mer peptides with residues starting at - 2, 4, 11, 18, 154, 162, 169, 171, 178, 199, 205, 209, 216, 241, 248, 250, 255, 262, 266, 267, 268, 273, 278, 280, 273, 286, 287, 298, 290, 309, 316, 332, 335, 350, 354, 358, 361, 371, 373, 384, 389, 397, 399, 400, 402, 407, 408, 420, 423, 425, 446, 449, 450, 456, 463, 465, 485, 488, 501, 512, 536, 544, 563, 570, 571, 576, 577, 578, 583, 585, 590, 592, 595, 598, 599, 601, 602, 603, 604, 606, 607, 613, 619, 648) and seventy-seven, 10-mer peptides with residues starting at - 9, 17, 57, 87, 96, 154, 161, 169, 177, 197, 199, 200, 208, 216, 224, 232, 240, 243, 250, 266, 267, 268, 272, 285, 287, 289, 297, 318, 323, 331, 342, 350, 355, 357, 365, 380, 383, 388, 391, 395, 399, 400, 406, 407, 409, 415, 432, 449, 453, 457, 462, 476, 484, 489, 492, 511, 519, 536, 543, 544, 548, 568, 570, 571, 576, 577, 584, 590, 595, 598, 599, 601, 602, 603, 605, 611, 629) were synthesized. Possible epitopes identified in the first screening were further purified by HPLC on a C-4 column (VYDAC, Hesperia, CA)(>98% purity) and the molecular weights of the peptides were verified by mass spectrometry measurement as previously described (Example 3; Kawakami, Y., et al., (1994) *J.Exp.Med.* 180:347; Kawakami, Y., et al., (1994) *Proc Natl Acad Sci (USA)* 91:6458).

Please replace the paragraph on page 97, line 31 through page 99, line 5 with the following:

With the exception of G10-4, which required a concentration of 1ug/ml to sensitize T2 cells for CTL lysis (Example 3; Kawakami, Y., et al., (1994) *Proc Natl Acad Sci (USA)* 91:6458), all gp100 epitopes identified in this study could sensitize T2 cells for CTL lysis at a concentration of 1ng/ml. G10-5 appeared to be inhibitory to the cytotoxic activity of CTL at concentration greater than 10ng/ml since lysis of T2 cells incubated with G10-5 at more than 10ng/ml was repeatedly lower than at 1-10ng/ml in this assay condition in which the peptide was present in the medium during entire 4h cytotoxicity assay. The relative binding affinity of these epitopes to HLA-A2.1 was also measured using an in vitro competitive binding assay (Table 13). $G9_{154}$ had an higher binding affinity (50% inhibition of the standard peptide at 11nM) to the HLA-A2.1 molecule than $G10_{154}$ (1010nM) which contains an extra leucine at the C-terminus of $G9_{154}$, and could sensitize T2 cells at lower concentrations than $G10_{154}$. $G9_{209}$ also bound to HLA-A2.1 with higher affinity (84nM) than $G10_{208}$ (2080nM) which contains an extra threonine at the N-terminus, and could sensitize T2 cells at lower concentrations of peptide than $G10_{208}$. Thus, the 9-mer peptides were superior to the corresponding 10 mer peptides in the sensitization of T2 cells to CTL lysis, and they also had higher binding affinities to HLA-A2.1. This was also the case for the identified MART-1 9 and 10 amino acid peptides (M9-2, M10-3, M10-4) (Example 2; Kawakami, Y., et al., (1994). *J.Exp.Med.* 180:347). The results of the peptide titration in the T2 cell lysis assay correlated with the results of the HLA-A2.1 binding affinity as measured by the in vitro binding assay. The other gp100 epitopes, $G9_{280}$, G10-4, or G10-5 had binding affinities for HLA-A2.1 with 50% inhibition at 95nM, 483nM, or 13nM, respectively. The HLA-A2.1 binding affinities of the previously identified HLA-A2 restricted melanoma epitopes in MART-1 (Example 2; Kawakami, Y., et al., (1994) *J.Exp.Med.* 180:347) and tyrosinase (Wolfel, T., (1994) *Eur.J.Immunol.* 24:759) were also measured (M9-2(397nM), M10-3(2272nM), M10-4(5555nM), $T9_1$ (333nM), $T9_{369}$ (40nM)). Except for the 10mer peptides ($G10_{154}$, $G10_{208}$, GM10-3, GM10-4), for which overlapping 9-mer epitopes ($G9_{154}$, $G9_{209}$, M9-2) existed, all melanoma epitopes had either high ($G9_{154}$, G10-5, $T9_{369}$) or intermediate ($G9_{209}$, $G9_{280}$, G10-4, M9-2, $T9_1$) binding affinities to HLA-A2.1.